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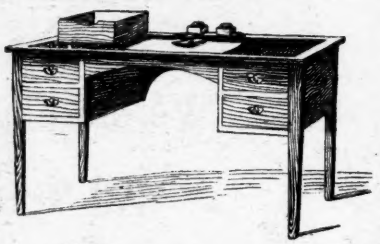

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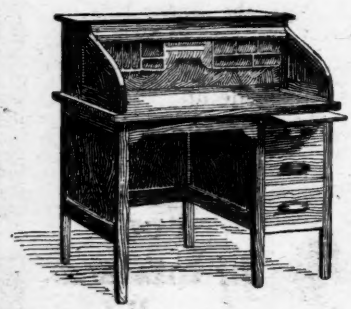

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PAPILLŒDEMA.[1]

By A. E. Mills, M.B., Ch.M. (Syd.),
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Papillœdema is the term which has in great measure and rightly so replaced the older name optic neuritis. Papillœdema means swelling of the optic papilla or, more accurately, an œdematous swelling of the papilla.

It is of various grades of intensity, varying from a scarcely measurable swelling of the papilla with slight engorgement of the veins, to a swelling so great that the papilla bulges forward; the veins become so engorged that they rupture, leading to clearly visible hæmorrhagic patches. The œdematous exudate obscures the papilla, which is now no longer definable, its situation being only recognizable from the fact that it is the meeting place of the venous branches coursing from the distal parts of the retina.

Such conditions are familiar to most of you. It would be well were they familiar to all, because papillœdema is a very common condition and a very serious one, arising from many causes. Although it is a common condition, it is very often unsuspected and as often unlooked for. Its recognition is of the highest diagnostic importance; without the knowledge of its existence many disease processes must pass undiagnosed. It is so serious that if it remains unrelieved, it will lead to blindness, due to the consequent optic atrophy. If it continues for any length of time it is bound to cause some deterioration in vision. Therefore I think I am not demanding too much when I ask that a knowledge of the normal and common abnormal appearances of the retina should be required of all medical students of our universities. I am glad to say that the proposed amendments to the curriculum of the Sydney Medical School will more specially require of our students a knowledge of the commoner diseases of the eye.

Although papillœdema is due to many causes—indirect causes—it is, in the main, part and parcel of a general intracranial œdema or, expressed more precisely, it is one of the results of increased intracranial tension; the subject of papillœdema cannot be discussed without a consideration of the broader question of increased intracranial tension.

Now, with regard to this question, there is much looseness of thought, as I gather from answers to questions put to advanced students. When asked: "What is the cause of increased intracranial tension?" I invariably get the answer: "Cerebral tumour"; but as cerebral tumour does not always produce this condition, it is obvious that little consideration has been given to the question and consequently an imperfect understanding of the matter exists in their minds.

We may well ask ourselves, firstly: "What is implied by the phrase 'increased intracranial tension'?"; secondly: "How is it brought about?" Let us consider very briefly some anatomical facts.

[1] Read before the Combined Sections of Medicine, Neurology and Ophthalmology of the Australasian Medical Congress, Brisbane, 1920.

(1) The skull, except in the infant, is an unyielding box. It contains within it the brain, with its membranes, the blood vessels, arteries, veins and venous sinuses and the cerebro-spinal fluid.

(2) There are no true lymphatic vessels in the brain and spinal cord.

(3) The brain itself is incompressible.

Normally every systole of the heart leads to an increased pressure within the cranium. During diastole the pressure is diminished. This is obvious to anyone who will observe the pulse movements of the fontanelle of a baby.

> The brain is an organ which, like any other organ of the body, pulsates with each stroke of the heart, but, owing to its peculiar condition, enclosed as it is within an unyielding box, the cardiac pulse is not entirely spent in distending the arterioles and capillaries, but is transmitted to the venous sinuses. The brain, expanded by the stroke of the cerebral arteries, expresses blood from the cerebral veins into the sinuses.—Leonard Hill.

Now the skull, an unyielding box, cannot be more than filled. Certainly the tension within it may be increased. This alteration in tension can only be brought about by variations in the fluid contents of the cranium, in other words, the blood and cerebro-spinal fluid.

Increase of intracranial pressure may be voluntarily brought about by attempts at forced expiration with the glottis closed, such as occurs when straining at a stool takes place. This increased tension is due to obstruction to the venous outflow from the cranium.

It may be objected that such an increase of blood pressure would be obviated by the displacement of the cerebro-spinal fluid from within the cranium to the more distensible spinal sub-arachnoid space. In this connexion we must remember that normally the amount of cerebro-spinal fluid is very small. Leonard Hill says: "Little more in amount than the synovial fluid in a joint." Be that as it may, it will be apparent to anyone who notices the bulging of the fontanelle of a baby during loud crying, that increased expansion of the brain, due necessarily to the increase in the amount of blood, does occur when there is an impediment to the venous return of blood from within the cranium to the heart. Such conditions as I have mentioned are only temporarily present and are within the range of physiological variations of pressure. But in conditions of sinus thrombosis, such as may occur in marasmic children or in the aged or at the close of infective diseases, such as pneumonia and enteric fever, there will be increased venous pressure. This increase of venous pressure will result in stasis and malnutrition and will lead to transudation of fluid, *i.e.*, œdema, in this case intracranial œdema, either local or general, dependent upon the sinus or sinuses involved.

But the question of intracranial œdema is not so simple. It is, I think, unphysiological and therefore incorrect, to treat the matter in such a way as to consider the variations of intracranial circulation and its results as something apart from and independent of the cerebro-spinal fluid. The cerebro-spinal fluid

is the secretion of the choroid plexuses or, as Mott expresses it, the "choroid gland." Now any gland depends for its functional activity upon a proper vascular supply. The choroid gland is not different from the other glands of the body in this respect. Nay, it resembles them in its wondrous selective action, for the fluid secreted by this gland is quite different from the blood which comes to it. It resembles closely, as Halliburton has shown, Locke's modification of Ringer's saline solution.

> The cerebro-spinal fluid is normally present at a certain pressure and this pressure is not the result of arterial pressure passively transmitted to the fluid. The cerebro-spinal pressure, it is true, may be affected by changes in the arterial and venous pressures, but it is not dependent upon them; and it may, and often does, vary quite independently of these. The true cerebro-spinal pressure is the result of the secretory pressure of the choroid endothelial cells. In other words, the cerebro-spinal fluid cannot any longer be regarded as a fixed quantity without the power of expanding and contracting in volume.—Halliburton.

We know that there are many conditions in which we find a large increase of cerebro-spinal fluid, an increase so great that the fluid spurts from the lumbar puncture needle in a continuous stream for some distance. But this increased amount of cerebro-spinal fluid is not necessarily an index of the amount within the skull. The spinal sub-arachnoid space is capable of considerable distension and if the choroid gland secretes fluid at a rate greater than the evacuant channels, *i.e.*, the perivascular and perineural spinal lymphatic channels, can remove, cerebro-spinal fluid accumulates. On the other hand, the tension of the fluid obtained by lumbar puncture is ordinarily an index of intracranial pressure. But increased amount of the cerebro-spinal fluid can arise only at the expense of the other fluid contents of the cranium and if long continued, at the expense of atrophy of the brain tissue. In infants under certain conditions it will lead to hydrocephalus.

Now the pressure of the cerebro-spinal fluid is never greater than the pressure of the arterial blood, otherwise it would not be possible for blood to enter the skull; but the pressure of the cerebro-spinal fluid may be so great as to hamper the circulation by impeding the venous circulation and deprive the brain of its proper supply of blood. When such a condition arises no great and immediate harm should result, for the increased tension of CO_2, which would necessarily ensue, would stimulate the vasomotor centre and bring about an increase of the general blood pressure. In fact, this does take place, as I have frequently demonstrated to my students. In apoplexy, for example, where there is a sudden escape of blood from a vessel, the patient dies, not from the extravasation of blood, but from the resultant grave anæmia.

> It is the effect of anæmia on the vital centres of the brain that kills.—Leonard Hill.

Soon after the hæmorrhage there must be great interference with the venous circulation and this must lead to the increased tension of CO_2, which stimulates the vasomotor centre so that an enormous rise of the general blood pressure takes place. This increase of blood pressure must cause a corresponding rise of intracranial tension.

But it may be asked: "Will not an intracranial tumour, a hydatid cyst, for example, cause increased tension purely and simply by virtue of its continuous growth within the unyielding cranium?" It is only by looking at the problem from all angles that we are likely to arrive at a correct conclusion. Well, in the first place a hydatid grows at the expense of the other intracranial contents; as it grows blood and cerebro-spinal fluid will be expressed from the skull and a certain amount of atrophy of the brain tissue will result. The effects will be an imperfect blood supply and the liberation of extract of brain substance. Imperfect blood supply will mean less oxygen and increased pressure of CO_2 within the blood vessels of the cranium. Now, "CO_2 and extract of brain substance are two of the greatest stimulants for the secretion of cerebro-spinal fluid" (Halliburton). Carbon dioxide is also the great stimulant of the vasomotor centre. The hydatid cyst then, as it grows, will bring about an increase of blood pressure and an increase in amount and in pressure of the cerebro-spinal fluid within the cranium. The tension of the cerebro-spinal fluid will indicate the increased intracranial tension. All these facts have a very important bearing upon the ætiology of papillœdema, as I will endeavour to show later on.

We are now in a position to answer the question with which we began the discussion. We mean by "increased intracranial pressure" an increase in the pressure of the fluid contents within the skull, an increase which may be dependent upon increased tension of the normal amount of cerebro-spinal fluid, but generally and in the vast number of cases upon an increased secretion of this fluid, together with an increased tension of it.

The second question: "How is increased intracranial tension brought about?" has already been partly considered, bound up as it is with the first question, but I will defer its complete answer to a later stage.

> Now it has been shown experimentally that there are three groups of substances which promote the flow and increase the pressure of cerebro-spinal fluid independently of those which affect it secondarily by altering the blood pressure.
>
> *First Group.*—Excess of CO_2 (or lack of oxygen in the blood), as in anæmia, and drugs which interfere with respiration.
>
> *Second Group.*—The volatile anæsthetics, which may act by interfering with respiration or by altering the physical conditions of secretion.
>
> *Third Group* is specific and consists of an extract of the choroid gland or of the brain. The specific stimulant in these extracts is of the nature of a hormone and probably acts directly on the secretory cells of the choroid gland.—Halliburton.

I have already very briefly considered the action of excess of carbon dioxide. The volatile anæsthetics do not concern us, but the third group demands our consideration.

> Now, in cases of general paralysis and brain softening, where katabolic processes are excessive, this hormone can be recognized by physiological tests in the cerebro-spinal fluid itself.—Halliburton.

In destructive and rapidly growing tumours of the brain substance we have the conditions for the liberation of this hormone and in such diseases we have, as a rule, an excessive amount of cerebro-spinal fluid under greatly increased pressure. It is in these cases

that we meet with the highest grade of papillœdema. I would suggest, therefore, that in rapidly growing, *i.e.*, destructive, tumours extract of brain tissue is set free in increased amount and acting on the choroid gland brings about the excessive amount of cerebro-spinal fluid which is so commonly found. And I would further suggest that the extract of spinal cord has a similar effect, for in cases of spinal tumour where an operation is undertaken for the removal of the tumour an enormous amount of cerebro-spinal fluid escapes immediately the dura and arachnoid are opened.

But it will at once occur to you that some cases of cerebral tumour are not attended with an increase of cerebro-spinal fluid. How can we explain this discrepancy? Let us bear in mind that some tumours grow rapidly, others grow slowly. The more slowly growing tumours bring about a slower destruction of brain matter. The amount of brain extract set free at any given time by such tumours will be much less than that set free by rapidly growing tumours causing great destruction of the brain. The greater the katabolic effect of tumours, the greater the amount of brain extract liberated to stimulate the choroid plexus and *vice versa*. It seems to me to be a question of mass action of this specific hormone, *i.e.*, brain extract, and nothing more. Certain it is that the size of the tumour has no relation to the amount of cerebro-spinal fluid secreted, for with a small tumour a large amount of fluid may be found and with a large, slowly growing tumour the amount of fluid may not be greatly increased.

I pass on now to consider substances, other than specific hormones, whose nature is unknown, but which produce an excessive amount of cerebro-spinal fluid. These demand our attention, for with the increase of fluid there may be definite papillœdema.

Perhaps it will interest you more if I quote actual cases.

Case I.—I was asked to see a little child in consultation. The history given was that the child had been suffering for many days from entero-colitis, with high temperature and raised pulse, that on the previous day general convulsions had developed and the child had then become comatose. There had been no evidence of disease of the lungs or kidneys. When I saw the child the coma was marked, there was considerable rigidity of all the muscles of the body with irregular slight convulsive movements more or less general. The question for consideration appeared to me to be: "What was the cause of the comatose state?" Was it due to poisons from the alimentary canal acting on the brain cortex producing abolition of its function, or was it due to compression of the brain resulting from increased intracranial tension? From close consideration given to many such cases and especially to the results which follow relief of tension, I have been forced to the conclusion that, while not denying the influence of toxæmia on the cortical cells, the main cause is mechanical pressure which in such cases is invariably the result of great increase in amount and tension of the cerebro-spinal fluid. Accordingly I suggested that lumbar puncture should be performed. This was done. Some 30 c.cm. of fluid were abstracted. The fluid was perfectly clear and apparently normal cerebro-spinal fluid. The rigidity and convulsive movements soon disappeared; after some hours the child became conscious and in a few days was practically well.

Case II.—A lady of 30 was seized with headache, which became very severe and caused vomiting. As the headache and vomiting became worse, a doctor was called in. She was told she was suffering from a bilious attack. Purgatives and sedatives were employed. The condition did not improve. Some three weeks after the onset of her trouble she came under my observation. Her sight was then very bad; she had to be led about. The headache was very marked and vomiting occasionally took place. Examination showed marked double papillœdema. I sent her to hospital, where lumbar puncture was done six times. The first four punctures resulted in the evacuation of a little more than 30 c.cm. each time. The remaining punctures produced 20 c.cm. and 16 c.cm.. The papillœdema gradually disappeared and the vision improved until it reached $^{6}/_{36}$ in one eye and $^{6}/_{60}$ in the other. Optic atrophy ensued and in consequence she had to resign her position as a teacher in the Education Department.

Case III.—A young man was sent to me by Dr. Brearley suffering from double papillœdema, with greatly defective vision. I was asked to see if I could find any definite cause for the condition. The only other symptom complained of besides the defective vision was headache more or less constant. My examination revealed that his urine was full of pus and motile organisms and that there was a stricture due to an old gonorrhœa. I sent him to Dr. Gordon Craig who gradually dilated his stricture; within a few days the urinary stream was normal, the pus had greatly diminished and the headache had disappeared. The vision improved from $^{6}/_{60}$ to practically normal in about six weeks.

Case IV.—A little girl, aged 3 years, was admitted on July 2, 1920, to the Royal Prince Alfred Hospital in a state of marked stupor. The mother stated that the child had not been well for three weeks. There was nothing definite; as the mother put it, the child seemed "off colour." At first the child had complained of earache, coming on at infrequent intervals and of short duration. Later the child had complained of some pain in the back of the neck and in the forehead. The drowsiness had come on a week before admission and had increased in intensity until it reached a state of marked stupor. There had been no vomiting, no convulsions, no discharge from the ear. The temperature on admission was 38.3° C., the pulse 120. There were some slightly enlarged cervical glands. The child lay huddled up in bed. Her neck was somewhat rigid, but there was no retraction of the head. The reflexes were normal and there was no evidence of paralysis. I attempted to examine the fundus; my efforts were quite in vain. A small fluctuating swelling was found in the scalp over the occipital region. This, on being opened, was found to be a suppurating dermoid cyst. With the little evidence before me I thought probably that the child was suffering from *encephalitis lethargica*; that the earache of which

the child complained, indicated that the child had had a sore throat; that this was the primary source of infection. Lumbar puncture was performed and 15 c.cm. of perfectly clear and apparently normal cerebro-spinal fluid under greatly increased pressure were obtained. Next day the child was better, sat up, played and conversed with the mother. The improvement continued for four days, when the stupor reappeared. On the fifth day after admission the child had a convulsion. Next day lumbar puncture resulted in 12 c.cm. of clear fluid being obtained. The stupor was less marked. The following day the child had another convulsion; lumbar puncture again performed; 10 c.cm. of fluid was obtained. Drowsiness again relieved, but the relief not so marked. Three days later the child had another convulsive seizure. By lumbar puncture 10 c.cm. of clear fluid were obtained, but there was little improvement. Next day the child died. *Post mortem* examination revealed an abscess beneath the tentorium, between it and the cerebellum, indenting and invading the left lobe of the cerebellum. There was no single, large abscess of the cerebellum, but there was a series of small, intercommunicating, suppurative foci throughout the substance of the cerebellum.

The source of the abscess was the suppurating dermoid cyst, for the channel through the bone extending from the cyst to the subtentorial abscess was found blocked by material that looked like inspissated pus. This cast of inspissated pus was actually withdrawn. This channel was evidently the pathway for a vein which probably had become infected and thrombosed.

From many points of view this case is very instructive, but I will especially refer to the fact that the abscess was only one factor in the causation of the increased intracranial tension and consequent stupor. The other factor was the increased secretion of cerebro-spinal fluid, for after the evacuation of fluid by lumbar puncture the stupor was greatly relieved. The case also shows that brain substance destroyed by a cerebellar abscess stimulates the choroid gland to increased activity.

From a great number of similar cases I have selected these to show that many substances of unknown origin cause the choroid gland to secrete fluid in excessive amounts and at a rate greater than the evacuant channels can dispose of in the same time. Necessarily the fluid accumulates and the tension increases. What can we say with regard to these unknown bodies? In case I. the cause was probably a toxin arising from bacteria within the intestine. In case II. I ascribe the illness to polio-encephalitis, or so-called X-disease, of which, in the district where the patient was teaching, other cases had occurred. Case III., I think, without doubt, was definitely associated with the pus and bacilli in the urine and the papillœdema and headache were directly due to the excess of cerebro-spinal fluid caused by toxins from the urinary tract.

Case IV. points, I think, definitely to the conclusion that brain extract liberated by the katabolic effect of the abscess was the factor in determining the papillœdema.

That infections generally can and do stimulate the choroid gland to increased activity I have proved from the results of lumbar puncture performed in many cases of pneumonia (especially when meningismus was present), influenza, without pulmonary complications, typhoid, entero-colitic conditions associated with stupor or convulsions, etc., cerebral syphilis, poliomyelitis (quite part from polio-encephalitis) and *encephalitis lethargica*. The fluid may be and generally is quite free from organisms or cells and closely resembles, if it is not identical with, normal cerebro-spinal fluid. I am not referring to cases of meningitis where the fluid obtained by puncture contains the specific organisms themselves and numerous leucocytes. In purulent meningitis probably the character of the fluid is due to the normal fluid *plus* an admixture of organisms and cells washed off from the pia-arachnoid. To me it seems that toxic substances from local infections in the throat, lungs, cord and other places, carried by the blood stream to the choroid plexus, act as stimulants to this gland, which secretes a fluid at a greatly increased rate, causing increased accumulation of fluid and increased tension.

No account of the causation of increased accumulation of cerebro-spinal fluid would be complete without reference to the observations by our colleague, Dr. Lockhart Gibson, on lead poisoning. He has demonstrated the frequency of lead poisoning in children in Queensland; he has shown how it arises; he has indicated the means by which it can be avoided and he has proved that lead causes great increase of cerebro-spinal fluid and papillœdema. In this connexion I wish to express my great indebtedness to Dr. Lockhart Gibson for much valuable information.

From the foregoing I think we are justified in answering the second question by saying that increased intracranial tension is brought about by brain tumours, brain abscesses and by increased accumulation of cerebro-spinal fluid. While a brain tumour or a brain abscess may be responsible for increasing intracranial tension, it must be borne in mind that they may also cause an accumulation of cerebro-spinal fluid. In such cases the increased intracranial tension is only in part due to the tumour or abscess; the other factor is the increased amount of cerebro-spinal fluid. In other conditions, diabetes, nephritis, meningitis, local or general infective states, trauma, lead encephalopathy, the one and only factor in producing increased intracranial tension is the secretion and accumulation of cerebro-spinal fluid.

In his interesting researches on the secretion and excretion of cerebro-spinal fluid, Weed has shown that by introducing a silk catheter into the third ventricle through the aqueduct of Sylvius the flow of fluid will continue for hours, probably until the secretory activity of the gland itself is exhausted. It has been demonstrated that such secretion can, by persistent stimulation, be prolonged and the rate of flow be much increased. Such an increase of fluid caused by the conjectural stimulants I have suggested, in the cases and conditions already mentioned, would occur. The pressure of fluid may be raised to such an extent that it would be forced into all the channels in connexion with the subarachnoid space. Now it is well known that such channels, patent and potential,

exist around the various cranial nerves and especially around that outgrowth from the brain—the optic nerve. But the space within the dural sheath surrounding the optic nerve differs from the space surrounding the other nerves in this important respect, that the dural sheath becomes continuous with the selerotic. Cerebro-spinal fluid, therefore, which may find its way into this space, is really in a *cul de sac* and if the fluid be under increased pressure, as it is in the numerous examples I have given, it must exercise its pressure on the structures contained within the sheath, namely, the optic nerve fibres, the central artery and vein and upon the so-called lymph channels which serve for the passage of waste products from the eye. Paton and Holmes (*Brain*, Vol. 33, p. 132) hold the opinion "that œdema of the papilla that constitutes tumour papillœdema, is in the first place due to the venous engorgement that results from the rise of the intravenous pressure that is necessary in order that the circulation should be maintained in the intravaginal portion of the vein where this is subjected to an increased sheath pressure. The increased sheath pressure is also the origin of the second factor, the obstruction to the lymph drainage from the papilla." Granting this, I maintain that the cause of the increase in intravenous pressure within the optic sheath is part of the general increase in intracranial venous pressure, brought about by the obstruction to the outflow from the cranium and that this obstruction is in the main caused by increased accumulation and increased tension of cerebro-spinal fluid from causes already mentioned.

But, as I have already indicated, there are certain conditions in which papillœdema is present and in which there is no evidence of a great increase in amount of cerebro-spinal fluid. I have in mind certain cases of cerebral tumour in which, on lumbar puncture, there was little increase in pressure of cerebro-spinal fluid, yet papillœdema was marked. Papillœdema in such cases must, therefore, be due to the increased tension of a normal amount of the fluid in the dural sheath obstructing the so-called lymph spaces or the cerebral veins or both channels. The increased intracranial tension must be ascribed to the growth itself. The absence of marked increase of cerebro-spinal fluid in such cases is probably due to the slow destruction of brain tissue, insufficient to stimulate the choroid gland to increased activity.

Treatment of Papillœdema.

If we recognize that papillœdema is, in the main, only an expression of increased intracranial tension for which there are many causes, then it should be our aim by exploiting every method at our command to try to find these causes. To this end we should endeavour to get as complete a history of the illness as possible and following on this to make a systematic examination of the organs of the body; nor must we be content with a mere examination of the heart, lungs, abdomen and urine. We must look for all those causes that yield those ill-defined toxic substances which, carried to the choroid gland, stimulate it to yield fluid at a rate greater than the evacuant channels can carry off, so that the fluid necessarily accumulates and may be the cause of the papillœdema.

If septic urine can yield a toxic body which can cause headaches and papillœdema, as I think case II., which I have already quoted, illustrates, I think we must search for all sources of septic absorption. We must examine the tonsils, the post-nasal space, the nose, with its accessory sinuses, the ears and the teeth. I have already expressed the conviction based on personal observation that infections generally can and do stimulate the choroid gland to increased activity. From this I would venture to suggest that the headache which so frequently accompanies the infections in their early stages is due or related to the rapid accumulation of cerebro-spinal fluid, as a result of increased activity of the choroid gland. Be that as it may, we must keep in mind when confronted with a case of papillœdema, that its immediate cause is increased tension of the cerebro-spinal fluid within the optic sheath, obstructing the venous and lymph flow, that this increased tension of the fluid is in the greater number of cases associated with an accumulation of the fluid; that the causes of the increased tension are the causes which promote increased secretory activity of the choroid gland; that these causes from the clinical point of view may be summarized as follows:

(1) Brain extract from katabolic effects of brain tumour, brain abscess or brain softening.
(2) Metabolic poisons, such as arise in the course of nephritis, diabetes, etc., leading to so-called albuminuric and diabetic retinitis.
(3) Lead, probably producing its effects in a similar manner to tumours.
(4) Toxic substances from local infective foci in various parts of the body.
(5) General infective processes.
(6) The anæmias.
(7) Meningitis, tubercular, syphilitic, meningococcal, influenzal, etc..
(8) Poliomyelitis, polioencephalitis and *encephalitis lethargica*.

The treatment of papillœdema, which may arise in and is one of the results of these various disorders, is necessarily the treatment of the disease of which the papillœdema is such an important and such a serious sign. In the case of local infections, *e.g.*, local purulent states, the removal of the pus and the infective agents will quickly bring about a cessation of exaggerated activity of the choroid gland. But in the meantime some relief of intracranial tension and papillœdema will be obtained by lumbar puncture, repeated, if necessary, daily. But to be really effective we must aim at withdrawing fluid from the cranio-vertebral subarachnoid space until pressure is reduced to normal limits.

This procedure may well be followed in those diseases where metabolic poisons, stimulating the choroid plexes, bring about increased intracranial tension and subsequent papillœdema, *viz.*, nephritis, diabetes, the grave anæmias, the various forms of meningitis and polio-encephalitis. Nay, I would go further and suggest that in those cases of infection of the gastro-enteric tract where there are signs of stupor or coma, with or without convulsions, lumbar puncture should be performed without hesitation.

The same applies to trauma of the brain, where there are signs of compression of the brain, *e.g.*, stupor and coma. It must, however, be borne in mind that lumbar puncture is contraindicated in those cases of increased intracranial tension in which the symptoms point to cerebellar tumour, for in a number of cases fatal results have followed this minor operation. I view the abstraction of cerebro-spinal fluid in all such cases as I view the removal of fluid from the pleura.

With regard to lead encephalopathy, we must seek to eliminate the lead by the urine and by the bowel. I have in mind the question of ionization which Dr. Lockhart Gibson has employed in such cases, but I have had no experience of its value.

With regard to brain tumours I would like to refer to the view expressed by Harvey Cushing that papilloedema is not to be looked upon as an early sign in such conditions. He takes credit, and rightly so, for having operated on three patients suffering from cerebral tumour in an early stage before papilloedema had developed. But as these patients reach us late, when papilloedema and other signs indicative of brain tumour are already marked, we must urge interference in order that the subsequent optic atrophy and blindness may be prevented. But, as Byron Bramwell has insisted, we must exclude the possibility of lead as a cause for the condition, for lead encephalopathy may closely simulate cerebral tumour.

We should have definite views as to the course to be pursued when the lesion is presumably of syphilitic origin. In this connexion I cannot do better than quote the judicious opinion of Harvey Cushing:

> In the presence or absence of a positive serum reaction or a definite history of lues, antisyphilitic treatment deserves only a brief, vigorous trial; if pressure and symptoms have been outspoken and do not become distinctly ameliorated in the course of a few days, a palliative decompression should be performed and the treatment subsequently resumed should the diagnosis remain in doubt. One valuable criterion of the effect of treatment and the need of early operation must rest with the condition of the eye grounds, which should be observed daily, for a choked disc may continue to advance, though subjective discomfort is lessened.

When we have a patient who exhibits papilloedema and who also presents other signs and symptoms indicative of brain tumour, it is our bounden duty to urge surgical interference for the relief of the increased intracranial tension. To quote Cushing once more:

> Surgical measures have two main objects in view: (*i.*) to expose and remove the growth, if that be possible; (*ii.*) to palliate the symptoms of pressure in case (*a*) of an impossible regional diagnosis, (*b*) of a lesion in a recognizable, though surgically inaccessible, region, (*c*) of irremovability on exposure, whether from excessive size, undue vascularity, or what not.

With regard to surgical palliative measures, I would urge that the opening of the skull should be of such dimensions that the brain may bulge sufficiently to compensate for the increased intracranial tension. A mere trephine opening is quite insufficient.

Discussing this aspect of the question with my friend, Professor J. T. Wilson, he suggested that relief of the papilloedema might be obtained by exposing the optic nerve within the orbit in such a manner as is done for the extirpation of a tumour of the optic nerve and removal of a portion of the optic sheath. Such a procedure would allow the cerebro-spinal fluid within the optic sheath to drain into the orbit, from which it would be removed by the lymphatics of that region. Further, seeing that the fluid within the optic sheath is in direct communication with the intracranial cerebro-spinal fluid, drainage of this fluid also would occur, with relief to intracranial tension. I commend this suggestion to the careful consideration of our surgical colleagues.

Reports of Cases.

BANTI'S DISEASE IN A CHILD.

By **Wolfe S. Brown, M.B., Ch.M. (Syd.),**
Honorary Medical Officer, Balmain and District Hospital, Sydney.

T.H., a female child of 9 years, was brought to my surgery on August 27, 1919, complaining of "constant colds, always being very pale and weak."

The only interesting point in the family history is that her father has been some years under my care with pernicious anæmia.

Physical examination revealed well marked pallor of the skin and mucous membranes and a spleen enlarged to about the level of the umbilicus. The other systems appeared to be normal and a blood examination revealed a well-marked secondary anæmia with leucopenia (see table). A diagnosis of Banti's disease was made and operation advised. She was meanwhile treated for one month with increasing doses of *liq. arsenicalis* and iron without improvement (see table).

Date.	Red Cells per c.mm.	Hb. Value.	Colour Index.	White Cells per c.mm.	Differential Count.	Remarks.
27.8.1919	2,400,000	30%	0.62	3,000	Neutrophiles 54% Lymphocytes 43% Eosinophiles 3%	No nucleated red cells
30.9.1919	2,100,000	30%	0.6	2,200	Not taken	Has been taking iron and arsenic
29.10.1919	3,300,000	42%	0.64	13,400	Neutrophiles 82% Lymphocytes 14% Eosinophiles 4%	Numerous megaloblasts and much anisocytosis
2.11.1919	3,900,000	48%	0.6	9,000	Neutrophiles 58% Lymphocytes 37% Eosinophiles 5%	No nucleated red cells
9.11.1919	4,050,000	56%	0.7	8,400	Neutrophiles 55% Lymphocytes 44% Eosinophiles 1%	No nucleated red cells
28.11.1919	4,200,000	60%	0.71	8,600	Not taken	
16.12.1919	5,000,000	82%	0.82	9,200	Not taken	
17.2.1920	5,000,000	84%	0.84	9,100	Not taken	
2.2.1921	5,100,000	98%	0.96	12,600	Not taken	

Sir Alexander MacCormick consented to do the operation and on October 27, 1919, at "The Terraces," under ether anæsthesia induced by the open method, removed a large spleen. There were few adhesions and no special difficulties seemed to disturb the operation, which was well borne by the patient.

For a few days after the operation numerous large nucleated red cells of megaloblastic type were seen in the blood films, but these soon disappeared. The spleen was uniformly enlarged. The patient made rapid and progressive improvement (see table); the improvement has been maintained up to the present time. There was no glandular enlargement either before or after the operation.

Reviews.

SOCIAL INEFFICIENCY.

We have received a monograph entitled "Intelligence and Social Valuation," by Professor R. A. Berry and Mr. S. D. Porteus.[1] It is described as "a practical method for the diagnosis of mental deficiency and other forms of social inefficiency." One of the chief objects of the authors is to facilitate the recognition of "potential social inefficients" among children of school age; these potential inefficients apparently include, as well as the mental defectives, many who are not mental defectives. "It is the dull and backward, the border-line cases, the mentally unstable and the physically and constitutionally inferior children who ultimately swell the ranks of the criminal, the vicious and the industrially incompetent."

The diagnosis is made from a consideration of the child's rating by the Binet and Porteus tests, the record of his height, weight, grip and vital capacity; his history as regards adaptation to home or school environment and the number and importance of the physical defects are taken into account. Tables of correlations between the physical and psycho-physical measurements and the intelligence tests are given. In addition, there are tables of correlations between Binet and Porteus test ages and industrial capacity and social fitness, as estimated by the School Principal and Industrial Director at the Vineland Training School.

It is stated that the Porteus tests disclose capacities not tested by the Binet method, such as prudence, forethought, planning capacity, ability to improve with practice and adaptability to a new situation. The authors combine the rating by the Binet-Porteus tests and adopt the average intelligence quotient 75 as the border-line of normality.

About one half of the monograph is devoted to a discussion on the relationship between intelligence and brain capacity, as estimated from head measurements. Much evidence is adduced to prove that a higher intelligence demands a larger brain and that, as in some mysterious fashion the growth of the brain governs that of the future skull, the larger brain necessitates a larger skull. Stress is laid upon the importance of the supra-granular layer of the cerebral cortex. The infra-granular layer controls the instinctive activities, is approximately as well developed in the higher animals as in man and has attained over 80% of its total adult thickness shortly after birth. The supra-granular layer, on the other hand, is the controlling layer and the layer through which education works; it has attained only 50% of its adult thickness at birth and shows an increase in development which is correlated with the educability and general intelligence which appear in an increasing degree during the ascent of the mammalian scale.

Percentile tables are given, which show what the brain capacity should be at each year of life from birth to twenty years and the deviations met with on either side of the median. The authors conclude that when the brain capacity is much below what their figure show it should be at any period of life, it is the supra-granular layer which is lagging behind in development and that, if this conclusion be confirmed by the other avenues of approach of their method, the diagnosis becomes certain.

In spite of the fact that estimations of the cubic capacity of the brain are liable to a not inconsiderable margin of error, the authors have shown the estimated capacity of brain in several abnormal groups to be considerably below that of normal groups of corresponding ages.

One of the principles on which the use of brain capacity in diagnosis is based is that, "as head growth is almost entirely governed by brain growth, striking deviation from the normal in head size will tend to be associated with mental abnormality," so that abnormality may be looked for among the unusually large-headed, as well as among the unusually small-headed. The authors propose to regard as being potentially abnormal only those individuals whose brain capacities fall below the 10 percentile and above the 90 percentile.

What is true of a class or race is not necessarily true of the individual; while the authors found that 50% of a small group of feeble-minded individuals had brain capacities falling outside these extreme percentiles, the other 50% fell within the limits regarded as normal. In them brain capacity could not be expected to be of much assistance in diagnosis and the degree to which development of the supra-granular layer has taken place would be obscured by failure of head measurements to disclose varying ratios of neuroglia, cerebro-spinal fluid and other contents of the skull. The intelligence tests were applied to 200 unselected persons whose brain capacity fell outside the extreme percentiles. Among these, 18.5% of the microcephalics and 5% of the macrocephalics (who apparently are not less numerous than the microcephalics) were classed "at feeble-minded levels," a frequency much greater than is to be found among the general population. On the other hand, 32% of the microcephalic children were only dull, while the remaining 49.5% were normal or above average intelligence. Among the macrocephalics 9% were dull, 61% normal and 25% above average intelligence. From these findings it is not altogether clear how, in the individual case, head measurement can give any clue to the development of the supra-granular layer of the cerebral cortex.

The rules for diagnosis indicate how the various parts of the method are to be applied. The authors regard children with an average Binet-Porteus intelligence quotient below 75 as potential social inefficients, as mental defectives who do not possess abilities making for social success. They assume that the intellectual retardation or the mental instability in such individuals is so grave as to render it most unlikely that they will ever make their way unaided in society. They regard children with Binet-Porteus quotients from 80 to 75 as on the border-line; the actual status of many of these could be determined by the application of their full examination method. "Children as dull as these who have, for instance, a brain capacity below the 10 percentile, must have restricted potientialities for further development, so that in their cases this evidence makes the diagnosis almost certain. Children who have a poor psycho-physical rating along with such a grave intellectual retardation, are also constitutionally inferior and are more unlikely, therefore, to earn their own livings."

The work done by the authors has necessitated the examination of thousands of children and the careful study of a great mass of data. There can be no question of the value for diagnostic purposes of the observation of characteristics which occur to a greater extent among the defective class than in the general population. A person who compares closely with the feeble-minded class and badly with the average individual when subjected to a sufficient number of these tests, may be justly suspected of being feeble-minded, but conduct, the person's reaction to the circumstances of real life, must be the deciding factor in the diagnosis of mental abnormality.

[1] Intelligence and Social Valuation: A Practical Method for the Diagnosis of Mental Deficiency and Other Forms of Social Inefficiency, by Richard A. Berry, M.D., and S. D. Porteus; 1920. Vineland, New Jersey: Published at the Training School at Vineland; Royal 8vo., pp. 100.

SANITATION AND HYGIENE.

The fact that a second edition of a book is called for within a comparatively short time after its first issue may be accepted as evidence that its contents have met with reasonable appreciation and that it has filled requirements in certain directions. "Elements of Hygiene and Public Health," by Dr. Jaising P. Modi,[1] has evidently met with such appreciation in India. The author states that it was

[1] Elements of Hygiene and Public Health: For the Use of Medical Students and Practitioners, by Rai Bahadur Jaising P. Modi, L.R.C.P. et S., L.F.P.S. (Glasgow), with an Introduction by E. J. O'Meara, F.R.C.S. (Eng.), D.P.H., I.M.S.; Second Edition, revised and enlarged; 1920. Calcutta, Sydney, etc.: Butterworth & Co., Ltd.; Demy 8vo., pp. 504.

written as a text-book to meet the special demands of Indian students and freely acknowledges the borrowing of subject matter from other text-books. This borrowing is almost inevitable, but unfortunately it has been carried out by Dr. Modi in such a manner as to impart an air of pedantry to much of the advice given and of impracticability to methods and measures described.

The statement (p. 14) that "enteric fever is more often spread through the medium of water than by any other means" cannot in the light of modern knowledge be accepted, although it is repeated with parrot-like persistency from text-book to text-book. An academic expression of opinion (p. 15) that water for drinking should not be stored in zinc receptacles is another example of an oft-repeated text-book fallacy. If, as indicated by the statement, such water be injurious to health, we should have had in Australia evidence of an overwhelming character. Zinc poisoning, however, is practically unknown here. The student is advised (p. 21) to see that cast iron water pipes are coated on the interior "with a glassy lining to prevent rusting." Most excellent advice, but how is the poor student to carry it out? Glass and enamel lined pipes have been introduced, but have been found altogether wanting in practice.

Text books might reasonably be expected not to lend themselves to the perpetuation of fallacies, such as that the addition of alum to water makes it hard. Yet we find (p. 27) that alum has the disadvantage of imparting permanent hardness to water. Standing by itself this may be absolutely correct, but the full case is not stated and the reader is not informed that the permanent hardness so imparted is at the expense of the temporary hardness and that the total is in no way affected. It is quite conceivable that under certain circumstances it might not be advantageous to transfer the hardness in this way, but when the amount of alum recommended (1 in 50,000) is considered, it must be recognized that the actual amount of hardness that would be affected is so small as to be practicably negligible under all circumstances.

In the part dealing with the analysis of water and air there are many errors and much misinformation. Castile soap is not soft soap (p. 33). The "oxygen consumed" in Tidy's test (p. 35) is usually accepted as that required to oxidize organic carbon, not nitrogen; it certainly cannot under any circumstances "correspond to the free ammonia in Wanklyn's test." In the test for nitrites (p. 35) we are told to use naphthylamine, when the α-naphthylamine should have been stated. The directions for the gravimetric estimation of lime, magnesia, etc., in water (p. 38) are absolutely meaningless and show much evidence of unintelligent borrowing.

The theory that on account of the CO_2 given off by trees at night (p. 44) it is dangerous to sleep near them, is novel and is certainly not supported by Australian experience. Some confusion of ideas is shown in the account of aqueous vapour in the air (p. 44). The air is stated to be saturated when it contains 50-70% of such vapour. The quantitative test for carbon monoxide in the air (p. 53) is to be made by absorbing it in a solution of copper sulphide. It is usual to employ an ammoniacal solution of cuprous chloride for this test and, further, no instructions are given for making a solution of the insoluble copper sulphide.

The illustrations are not good; many are reproductions without acknowledgement from other publications, while some that are obviously original, are grotesque absurdities, *e.g.*, that of a latrine on p. 211 and of a water closet and cistern on p. 223.

In carrying out the hydraulic test for drains, we are told (p. 233) to "plug the distal end of the drain with a rubber cork after filling it with water." One would have to be an Indian juggler to carry out this manœuvre!

The book has absolutely no use outside India and even to be of real and sound service there requires much revision and correction.

Naval and Military.

APPOINTMENTS.

The following appointments, etc., have been announced in the *Commonwealth of Australia Gazette*, Nos. 10 and 11, of January 27 and February 3, 1921.

Australian Imperial Force.

APPOINTMENTS TERMINATED.

Second Military District.

Captain A. L. Maclean, 25th December, 1920.

Third Military District.

Major S. T. Appleford, 21st April, 1919.
Captain H. W. Franklands, 2nd August, 1919.
Captain R. N. S. Good, 20th November, 1918.

Fourth Military District.

Lieutenant-Colonel H. C. Nott, 18th December, 1920.

Australian Military Forces.

Third Military District.

Reserve of Officers—

The undermentioned officers to be placed on the Retired List, with rank as stated, and with permission to retain such rank and wear the prescribed uniform, 1st January, 1921:

With rank of Colonel—Colonel F. D. Bird, Honorary Colonel Sir H. C. Maudsley, K.C.M.G., C.B.E..

With rank of Honorary Colonel—Lieutenant-Colonel N. B. Gandevia, V.D., Honorary Colonel G. A. Syme, Honorary Lieutenant-Colonel A. Honman.

With rank of Lieutenant-Colonel—Honorary Lieutenant-Colonel J. W. Springthorpe.

With rank of Honorary Major—Captain O. G. Taafe, Captain (Honorary Major) C. L. Lempriere.

The undermentioned Honorary Majors to be Majors—

John Thomson Tait, Maurice Buchan Johnson, William Walter McLaren, David Moore Embleton, O.B.E., Francis Tuelon Beamish, O.B.E., Andrew Joseph Brenan, Alfred Fay Maclure, O.B.E., Hildred Irving Carlile, William Alexander Morton, Harrie Bertie Lee, D.S.O., M.C., Arthur Hunter Powell, D.S.O., George Seaborne Robinson, M.C., Joseph Horace Downing, William Henry Rennick, James Vine Pearce, Valentine Francis Crowe, Colin Coape Simson, M.C., Eric Ivo Lowther Graves, James Iver McIver Chirnside, M.C., John Sandison Yule.

The undermentioned Honorary Captains to be Captains—

Roy Fallowes Watson, Frank Payne Edwards, Mark Aloysius Ley, John Alexander Shanasy, M.C., Patrick Shaw, Alfred Plumley Derham, M.C., Robert Leslie Forsyth, John Gratton Wilson, Ewen MacKenzie, Samuel Leslie Germon, James Warne, James Ignatius Rowan, John Wilkie Young, George Sutton, Gordon John Kennedy McIver, David Dorey Brown, George Vernon Davies, D.S.O., Cyril Florance MacGillicuddy, William Rock, Stewart Osburn Cowen, William Henry Orchard, M.C., Francis George Travers Champion de Crespigny, Alexander Allison Lang, Thomas Simpson Campbell, Rayner Laming Bellamy, Valentine Macdonald, Reginald Patrick MacGillicuddy.

Fourth Military District.

Australian Army Medical Corps—

To be Lieutenant-Colonels—

Major S. R. Burston, C.B.E., D.S.O., and Captain (Honorary Lieutenant-Colonel) C. Yeatman, O.B.E., 1st July, 1920.

Fifth Military District.

Australian Army Medical Corps Reserve—

To be Honorary Captain—

Stanley Earles Craig, 1st December, 1920.

Honorary Captain F. C. Thompson to be transferred to the Australian Army Medical Corps Reserve, 2nd Military District, 1st January, 1921.

The Medical Journal of Australia.

SATURDAY, FEBRUARY 26, 1921.

The Cerebro-Spinal Fluid.

Among the subjects selected for special discussion at the Australasian Medical Congress, Brisbane, 1920, was that of papillœdema. It was recognized that much confusion and little precise knowledge were responsible for faulty teaching and haphazard practice in connexion with the pathological conditions associated with "choked disc." A full debate is one of the best means of focussing the attention of the medical profession on an imperfectly understood problem and of guiding fresh research by differentiating between proven facts and unfounded hypotheses. The Executive Committee of the Congress were wise in their selection of this subject for discussion and wise in the selection of Professor A. E. Mills as the first speaker. The debate proved disappointing, notwithstanding the excellent paper by Professor Mills, which will be found in this issue. While Professor Mills adopted the orthodox and quite essential course of introducing the subject by defining the term, many of the subsequent speakers perpetuated the existing confusing by failing to distinguish the œdematous swelling of the papilla from inflammatory conditions of the same structure. The only other speaker who discussed the mechanism involved in the production of an œdematous swelling of the disc, was Dr. R. R. Stawell. The fundamental proposition in the teaching of Professor Mills is that papillœdema is produced by the same causes that lead to an increased intracranial tension. The actual congestive condition of the papilla can be dismissed without misgiving. Compression of the veins and lymphatics in the sheath of the optic nerve results in a congestive hyperæmia. It is therefore necessary to consider the causes of this compression in order to arrive at an exact knowledge of the nature and significance of this sign. Dr. Stawell has suggested that when the cerebro-spinal fluid is prevented from passing from the subarachnoid space into the vaginal sheath, papillœdema cannot arise. If the obstruction to the passage of the cerebro-spinal fluid involves a compression of all the vessels, the result would necessarily be an anæmia and not a hyperæmia of the disc. Professor Mills analyses the cranial contents with a view to the determination of those elements that are capable of variation in quantity and variation in density. The mass of brain and its membranes are of constant volume. Strictly speaking they are compressible under definite physical conditions. For the present purpose, however, they may be regarded as practically incompressible. In a similar manner the cranium may be regarded as a rigid, unyielding cavity, although this is not absolutely true. The amount of blood and of cerebro-spinal fluid within the cranium at a given time varies considerably. Professor Mills has presented all that is known concerning the variations of the quantity of cerebro-spinal fluid secreted and the variations of the intra-cranial pressure induced by changes in volume of the fluid within the cranial cavity. On this clear and succinct summary, he has offered an explanation for the condition of increased intra-cranial tension. His hypothesis is so plausible and so admirably fitted to the ascertained facts that we need have no hesitation in accepting it tentatively. It serves as a safe working hypothesis both for clinical diagnosis and for further investigation. Moreover, it helps us to analyse the physical conditions within the skull in any patient exhibiting the sign of papill-œdema and therefore serves a very useful purpose.

There are certain points still requiring elucidation. In the first place we may assume that the space available for the cerebro-spinal fluid is considerably greater than that habitually used. Under ordinary conditions when an excess of fluid is excreted by the choroid gland, the additional volume is temporarily stored in the spinal segment of the subarachnoid space. It is unknown what are the exact physical conditions that govern the relations between the tension of the spinal fluid, the cerebral fluid and the blood. This subject has recently been approached and some of the phenomena studied, but so far there does not appear to have been any definite endeavour made to register continuous pressure curves when the blood pressure is being depressed or raised by artificial means. The second point needing investiga-

tion is the mechanism governing the rate of escape of the cerebro-spinal fluid into the peri-vascular and peri-neural spinal lymphatic channels. Nature has, as a rule, a wonderful knack of introducing a compensatory mechanism by means of which a balance is maintained between secretion and excretion, an automatic regulator system. Such a safety valve action breaks down only when a mechanical defect occurs or when by overstimulation, the glandular activity is increased beyond the safety limit. Professor Mills reminds us of the willingness of the choroid gland to work overtime and at high pressure. We should endeavour to find out how much extra work can be performed without danger to the individual.

One part of Professor Mills's hypothesis seems to be speculative. Halliburton showed that in the disintegration of brain tissue a substance is liberated which has the function of stimulating the choroid gland to greater activity. He escapes from the difficult question of the nature of this function by postulating a hormone. Definite evidence is available in regard to the action of carbonic acid on the choroid gland. For the present we must be satisfied with the knowledge that extracts of disintegrating brain substance have the same effect. Professor Mills goes further. He has observed the signs of an increased intra-cranial tension in persons suffering from diverse pathological conditions. To meet the difficulty he suggests an unknown toxin. Until the actual existence of a toxic substance liberated in solution from a focus of septic or other infection has been proved, this speculation must be accepted with reserve. In the case of the few bacteria which give rise to true exo-toxins, such as the diphtheria, the tetanus and the dysentery bacilli, a specific action on the choroid gland has not been described. In the case of plumbism, it is known that the heavy metals produce primarily a degenerative effect on the endothelium of the capillaries. Special tissues exert a considerable degree of selective affinity for one or other metal. All tissues, however, are occasionally attacked by any of the heavy metals. No doubt the choroid gland is at times affected by lead. The result would be a diminution of the glandular activity. When lead leads to increased secretion of cerebro-spinal fluid, the attacking point is not the choroid gland. It may be the kidney, for the result, as it well known, of destruction of the renal parenchyma is an obstruction to the renal circulation and consequently a raising of the general blood pressure.

The lesson which the general practitioner should learn from this debate is that it is extremely important to recognzie papillœdema early and to work along the lines indicated by Professor Mills.

THE MECHANISM OF HYPERSENSIBILITY IN MAN.

When Richet published his first studies on the increase in toxicity of extracts of actines on repeated injection in dogs and suggested for the phenomenon of artificially induced hypersensibility to an organized substance the term anaphylaxis, it was assumed that the process depended on some form of biological response—the production of an antibody as a result of the introduction of an antigen. Richet's work dates back as far as 1898 and must be regarded as the fundamental studies on which the present teaching of anaphylaxis is based. Seven years later von Pirquet and Schick published a very valuable book entitled "*Die Serumkrankheit*" (serum sickness) in which they sought to identify with anaphylaxis the mechanism responsible for the signs and symptoms of the disturbance frequently observed soon after an injection of antitoxic serum. Much confusion existed during the early years of the present century as a result of a lack of limitation to the application of the term anaphylaxis. This confusion was not lessened by the recognition of unusual or occasional toxic symptoms following the injection of proteid substances into animals and man. The introduction of the terms "immediate reaction" and "late reaction" naturally led to a complete change in the significance of the phenomenon concisely and clearly described by Richet. Richet's experiments dealt with an increase of toxicity following a preliminary injection of the proteid substance. Rosenow and Anderson showed that the injection of extremely minute quantities of serum in guinea-pigs sufficed to render these animals anaphylactic. It was therefore difficult to accept the views held by many workers that this process of artificial hypersensibility to a protein was identical with the spontaneous production of signs of poisoning following the first injection of a protein. Paul Ehrlich taught that antibody production was not possible save when an organized substance was used as an antigen. It was therefore only adding to the confusion when an endeavour was made to include among the alleged anaphylactic phenomena the curious hypersensibility exhibited by certain persons to drugs like cocaine, iodoform and arsenic. In order to bring the various conflicting views into consonance von Pirquet evolved his theory of allergy. This condition arises when the normal or usual power of reaction of the organism to an organized substance is altered. This alteration may be either an increased susceptibility, a hyper-

sensibility or a diminished susceptibility. The idea of the allergic state satisfied von Pirquet and many other investigators as an explanation of the symptoms of serum sickness. The power of reacting was increased in the individual and consequently the introduction of usually non-toxic antiserum resulted in a flare up, an exaggerated response. The term is convenient, but very vague. It is quite unknown what mechanism is concerned in the alteration in the animal organism of the power to react to an antigen. In anaphylaxis it has been shown that an amboceptor is formed in the tissues of the guinea-pig as a result of the introduction of small quantities of a protein. Friedberger calls this amboceptor, when combined with the antigen, "anaphylatoxin." The amboceptor or antibody mixed with the protein employed as antigen and fresh complement is capable of killing normal guinea-pigs. It is therefore evident that von Pirquet and Schick were inconsistent in claiming at the same time that serum sickness was an anaphylactic phenomenon and that it depended on an allergic state. Moreover, everyone who has work at this subject, has discovered afresh that the different species of animals react in a varying degree to the initial injection of the proteid substance. While guinea-pigs are extremely susceptible, it is impossible to obtain a definite anaphylactic shock in monkeys. Rabbits respond in a milder manner and a fatal anaphylactic shock is but rarely observed in these animals. Dr. Arthur F. Coca has thrown up the question for discussion whether anaphylaxis occurs at all in human beings.[1] He argues that true anaphylaxis is characterized by typical symptoms varying according to the species when the same group of substances is used, by uniformity of symptoms in any one species for various substances and by the appearance of symptoms different from those normally produced in the physiological action of the substance. The first two qualities are undoubtedly characteristic of anaphylaxis. The third quality, however, does not appear to conform to all the phenomena. In Richet's experiments with actino-congestin and mytilo-congestin the symptoms following the repeated doses were merely those of increased physiological action. Dr. Coca shows that the tuberculin reaction, which is held by many to be an instance of anaphylaxis, is manifested by the same symptoms in all species of animals. He also claims that since the symptoms of toxin hypersensibility are not different from those of the normal physiological action of the toxins employed, they cannot be regarded as evidence of anaphylaxis. He points out that in human beings hypersensibility is nearly always limited to a particular substance in one individual, but the symptoms are the same in different individuals for a large variety of substances. Human hypersensibility is often inherited, but anaphylaxis cannot be inherited, although it may be transmitted from mother to fœtus *in utero* through the maternal circulation. He gives a comparison between the desensitization in the anaphylactic animal and the acquisition of tolerance in the human subject. In the latter cutaneous tests are capable of demonstrating a continuance of the hypersensibility. These and other considerations have induced him to regard the phenomena of hypersensibility in human beings as manifestations of allergy and not as indications of anaphylaxis. While there is much that cannot be gainsaid in his arguments, it is unlikely that his views will receive universal acceptance until the nature of the allergic state can be more accurately defined. At present we know as little concerning allergy as we know concerning drug idiosyncrasy. Neither really explains the phenomenon of hypersensibility and neither brings us nearer the solution of the problem of the proper method of attacking serum sickness and other specific instances of hypersensibility in man. It would seem that little could be gained by the attempt to find a single hypothesis to cover all these different manifestations. They have one element in common, namely, that the individual responds more violently than usual to a substance possessed of a definite physiological action. On theoretical grounds we are inclined to agree with Dr. Coca that anaphylaxis in the sense in which Richet used the term, does not occur in man.

[1] *The Journal of Immunology*, July, 1920.

JOHN KEATS.

On Friday, February 23, 1821, there died of consumption in a little tenement of the Piazza di Spagna in the Eternal City John Keats, poet and physician, Nature's nursling, "who grew like a pale flower by some sad maiden cherished." The son of a stable-keeper, he was born in London in 1795. The auguries for his future were good, for he was prematurely born and entered this world mantled in a caul. In 1814 he was admitted to Guy's Hospital as a student of medicine and showed surprising diligence at his work. He graduated in July, 1816, passing his final examination with credit, and for a short time practised his profession. He is even said to have performed several feats of surgery, but soon the genius of poetry beckoned to him and he became the immortal singer of green fields and blue skies. On this centenary of his death the profession loves to honour his memory, for he was the only great poet of England who was, for a time at least, a good practitioner of medicine.

The attention of our readers is directed to the increase in the price of this *Journal* to 1s. per issue. The Directors at their last meeting were reluctantly compelled to order this increase in price on account of the high cost of production and of paper. The subscription rate for new subscribers will be £2 for Australia and £2 5s. for abroad. The increase will not affect members of the British Medical Association, who receive *The Medical Journal of Australia* as one of the privileges of membership.

Corrigendum.

A typographical error occurs on page 163 in our issue of last week, which calls for correction. In the summary of the proceedings of a meeting of the Queensland Branch of the British Medical Association, at which the amendments to be introduced into the uniform lodge agreement were discussed, the date on which the amended provisions are to be introduced was given as April 1, 1912. The date should read April 1, 1921.

Abstracts from Current Medical Literature.

THERAPEUTICS.

(78) Adrenalin and Chloroform.

W. J. R. Heinekamp has made some experiments upon the modification produced in the action of adrenalin upon the heart by chloroform (*Journ. Pharm. and Exper. Therapeutics*, November, 1920). It has been known for some years that the injection of adrenalin into dogs, anæsthetized with chloroform, not infrequently causes a fatal result. The previous division of the vagi does not affect the result. As a rule large clots are found in the heart and great veins. The author has carried out a series of experiments upon dogs in which he has examined the effects of the injection of adrenalin and the inhalation of chloroform. The dogs were anæsthetized with ether. As is well known, chloroform is toxic for heart muscle, giving rise to a diminution in the force of the muscular contraction. If chloroform is administered after an injection of adrenalin, the rate of heart beat is greatly diminished, and complete inhibition may occur. This is due primarily to a paralytic dilatation of the heart on which ventricular fibrillation may supervene. This action is peripheral, since it occurs after section of the vagi, or after the administration of the atropine. The author is of opinion that the use of adrenalin is contra-indicated whenever chloroform has been employed and, further, that chloroform should not be administered whenever adrenalin has been used.

(79) The Carminative Effect of Volatile Oil.

O. H. Plant has studied the effect of volatile oils on the muscular movement of the intestines (*Journ. Pharm. and Exper. Therapeutics*, November, 1920). The opinions expressed in various text-books suggest that a difference of opinion exists as to the type of change produced by therapeutic doses of volatile oils on the muscular movements of the alimentary canal. Some authors say that volatile oils cause relaxation of the gastric and intestinal musculature and this relaxation allows the escape or onward progression of gases or contents passively. Others state that carminatives increase the muscular movements of the intestine. The author points out that the dose of volatile oil is small, so that the effects may easily be obscured by experimental conditions when such disturbing factors as anæsthesia and operative procedures, are present. A study has, therefore, been made of the effects of volatile oils on the intestinal movements of animals, in which no anæsthetic has been employed, and in which no operative proceedings have been necessary at the time of the experiment. The experiments have been carried out on two dogs in which isolated loops of the intestine have been brought up to the surface of the abdomen. The loops have been about 50 cm. in length. The skin wounds have healed slowly, so that a month or so has elapsed after the operation before any experiments could be carried out. The movements of the isolated loops have been recorded by means of a balloon attached by a rubber tube to a bellows recorder. The balloon and the rubber tube have been filled with water, so that a pressure of about 25 cm. of water has been exerted on the loop. The dogs have been trained to lie down quietly and they usually have slept throughout the experiments. The volatile oils have been applied either as the official "water" or after dilution of the "water" with several volumes of water. The great majority of the experiments have been performed 18 to 20 hours after feeding, but a few experiments have been carried out two or three hours after a meal. Altogether, 55 sets of observations have been made, in which more than 100 applications of volatile oils have been made. The primary effect of an application is an increase in tone of the intestinal wall, as shown by a rise in the level of the tracing, and by an increase in the extent of the rhythmic tracing. During the period in which the tone is increased, progressive contraction rings of peristalsis may be noted. Occasionally this increase in muscular activity is followed by a decrease in tone and by a diminution in the amplitude of the rhythmic contraction. These effects of the volatile oils are lessened, but are not abolished by the previous administration of atropine. They are completely abolished if the loops of intestine are washed out with a dilute solution of cocaine. When the muscular activity of the intestine is increased by the injection of a small quantity of morphine, the volatile oils produce the same increase in tone and augmentation of rhythmic contraction as in the normal animal. Although the experiments are not of a character to make evident quantitative differences, it is possible to arrange the results into two groups which merge into one another. Thus a group of volatile oils, including oil of mustard, cinnamon, peppermint, cloves, nutmeg, spearmint, juniper and lavender, as well as menthol and camphor, produces a more pronounced effect in dilute solution than the group of oils comprising oil of anise, cardamons, fennel, orange and carraway.

(80) Elimination of Carbon Monoxide.

Tandell Henderson and H. W. Huggard have made some studies upon the rate of elimination of carbon monoxide from the animal body and make some suggestions on the line of treatment to be adopted in those persons who have experienced a dangerous degree of asphyxiation as a result of the inhalation of carbon monoxide (*Journ. Pharm. and Exper. Therapeutics*, August, 1920). Within a few hours after profound but not fatal poisoning no trace of carbon monoxide can be found in the blood, but for days and months structural degenerations and functional derangements, either nervous or cardiac, may continue. The authors have studied the conditions under which CO is removed from the body and suggest a line of therapy or more correctly prophylaxis in the care of persons who have been poisoned with CO. During the development of asphyxia due to carbon monoxide there is vigorous hyperpnœa. Thereafter there is diminished production of carbon dioxide, probably owing to deficient oxygenation. Owing to the deficiency in carbon dioxide in the blood animals asphyxiated with carbon monoxide exhibit a great depression of breathing when restored to pure air. The rate of elimination of carbon monoxide is correspondingly slow. The condition of asphyxiation of the tissues thus continues, although the body is surrounded by pure air. The authors suggest that this post-gassing period is of critical importance in producing subsequent degenerations and derangements. Its abbreviation is an important object in suitable therapy. The inhalation of oxygen is of little value, as is well known from practical experience. The experiments show that it is not adequately inhaled. The inhalation of carbon dioxide diluted with air immediately augments the respiration and increases the elimination of carbon monoxide. The inhalation of mixtures of oxygen and carbon dioxide is even more effective. The increased breathing enables the oxygen to displace rapidly CO from the blood. Functional restoration is correspondingly accelerated.

(81) Carminative Action of Volatile Oils.

J. W. C. Gunn (*Journ. Pharm. and Exper. Therapeutics*, August, 1920) has endeavoured to ascertain the mode of action of volatile oils as carminatives. These bodies diminish feelings of distension and discomfort in the stomach usually with eructation of gas and allay or prevent griping in the intestines. The author recorded the movements of isolated strips of intestine from the rabbit, rat and cat according to the method of Magnus. The volatile oils were shaken into an emulsion with saline solution and measured quantities of this emulsion were added to the baths. The action of camphor, and of oils of anise, carraway, cassia, cinnamon, cloves, fennel, peppermint, rosemary and turpentine were tested. All decreased the rate and extent of the movements of the muscular strip and gave rise to relaxation. The action was reversible on replacing normal saline solution in the bath. Experiments were also conducted on the intestine *in situ*. Small amounts of emulsions were introduced into the intestine through a fine canula and the results recorded graphically. The animal was kept in a bath of warm salt solution. In many cases relaxation of the intestines was brought about. Similar observations were made on the stomach *in situ*. The author considers that the carminative action can be explained by relaxation and inhibition of movement in plain muscle.

UROLOGY.

(82) Fulguration in Residual Vesical Tuberculosis.

A Parisi (*Journal d'Urologie*, May, 1920) reports the treatment by fulguration of vesical tuberculosis which persists after removal of a diseased tubercular kidney. The vesical disturbance is diversely influenced by removal of the renal focus. In the great majority of cases there is cure or definite improvement of the pain and frequency, but sometimes mild relapses occur. In a few cases the cystitic symptoms remain, but the general health improves. Finally in very rare cases the cystitis appears to be aggravated by the operation. Marion states that the incidence of residual cystitis is far greater in poor patients, thus demonstrating the value of general treatment in the cure of vesical tuberculosis. The factors which determine the residual cystitis after nephrectomy for tuberculosis are: (*a*) The degree of the cystitis before the operation; even nowadays practitioners treat chronic cystitis as an entity, thereby not only allowing it to advance since the primary focus is untreated, but often adding secondary infection to the tuberculous lesions. (*b*) The influence of the ureteral stump, as it has been shown that the stump may keep on discharging tuberculous pus into the bladder for some time; in severe cases the whole ureter should be removed as well as the kidney. In less severe cases, ligature and division of the duct a short distance below the kidney gives good results. It is suggested that where the presence of a ureteral stump is suspected, its interior should be destroyed by the fulguration point applied with the aid of the cystoscope. In cases of uncomplicated tuberculous infection, the lesions in the bladder are simpler than those observed before the nephrectomy and usually consists of ulcers. These are of three types: (*i.*) typical shallow pseudo-ulcers, with irregular edges and practically no loss of substance; (*ii.*) true ulcerations, with excavated white or yellow floors; and (*iii.*) ulcers causing much loss of substance, with a red granular floor dotted with pus shreds or calcareous salts. These lesions are very frequently situated near the apex of the bladder and the author believes that the frequency of basal and peri-ureteral ulceration has been exaggerated. He quotes Marion's dictum that the presence of *ulcération apexienne* in the bladder is diagnostic of renal tuberculosis. The foregoing lesions are those of uncomplicated tuberculosis and are accompanied by mild symptoms. When, however, secondary infection is superadded, the symptoms are greatly aggravated and the cystoscopic appearances are multiform (ulcerations, general cystitis, vegetations, diphtheroid membranes and bulbous œdema). The bladder capacity may be reduced to 30 c.cm., rendering cystoscopy almost impossible. Heitz-Boyer was, in 1914, the first to use high frequency currents in the treatment of vesical tuberculosis. He used true fulguration, *viz.*, an attack by sparks produced from an electrode not in actual contact with the ulcer. The author prefers it to Beer's method of electro-coagulation by actual contact, as in cases of ulceration the thinned bladder wall may be liable to perforation. The apparatus employed is that of d'Arsonval-Griffe, which, though constructed for diathermy (electro-coagulation by contact), gives sparks powerful enough to destroy bladder tumours and, *à fortiori*, tuberculous ulcers. General anæsthesia is deprecated. Local anæsthesia of the bladder is used by means of a 4% cocaine solution acting for five minutes. The platinum electrode is held near enough to the ulcer for sparks to pass. In a few seconds the part of the ulcer immediately opposite the electrode is blanched, when the application is shifted to neighbouring points until the whole surface is whitened. The sittings should be not less than a fortnight apart and sedative suppositories are used for a few days after the fulguration. Hæmorrhage does not occur, but occasionally a reaction shown by pain and increased frequency persisted for several days. Success was not attained in all cases. The best results are seen in "ulcerative vesical tuberculosis"; that is, where no general cystitis exists in addition to the purely tuberculous ulcers. In cases of "tuberculous cystitis with diffuse lesions" injections of lactic acid bacilli or various irrigations and instillations give the best results; but even in these cases the ulcerations in the midst of the diffuse cystitis may be advantageously treated by fulguration. Twenty-one cases are reported in detail. Ten patients were cured and nine showed distinct improvement. The remaining two were uninfluenced by the treatment.

(83) The Action of Hexamine.

A. T. Shohl and C. L. Deming (*Journal of Urology*, October, 1920) investigated some of the problems of the action of hexamine. There are still many gaps in our understanding of the action of this valuable drug and of the best methods of administering it. These problems were investigated by means of various experiments: (1) Concentration of formaldehyde necessary to kill *Bacillus coli* in urine. The urine should be as acid as possible to secure an adequate conversion of the hexamine into formaldehyde and ammonia. The time of action is as important as the concentration. Hexamine was added to sterile fresh urine to make various concentrations, and bouillon cultures of *B. coli* were exposed to the action of this urine for varying periods. A concentration of 1 in 5,000 killed 67% of the organisms in 15 minutes, 86% in 1 hour and 100% in 24 hours. A concentration of 1 in 20,000 kills 50% in 2 hours, but the remainder grow normally after this time, and quickly replace the killed germs. So the supply of formaldehyde should be renewed throughout the day to ensure killing off. (2) Percentage of the hexamine split up at varying acidities. Patients received one gramme of hexamine four times a day and the 24 hours' specimen was tested. The acidity of the urine was measured by estimating its hydrogen ion concentration. With a distinctly acid urine (pH 5.0) 20% of the hexamine given in the 24 hours was split up, 13% with a less acid urine (pH 5.4) and none at all in distinctly alkaline urine (pH 7.6). (3) Loss of hexamine in the stomach. Unless the kidneys are damaged, 60% to 75% of the hexamine given is excreted by these organs. The remainder is excreted in small amount by the saliva and gall bladder, but it is mainly lost by conversion into formaldehyde by the gastric juice. This loss may be lessened to a great extent by following Thomson Walker's practice of giving the drug in large quantities of water between meals. (4) The effect of large fluid intake. Henderson and Palmer showed that the more concentrated urines are the more acid. Thus if fluids are given in large quantities to aid the renal function which is impaired, acid should be ingested and, when the urine attains the proper reaction, hexamine should be exhibited in increased quantity. In order to test the laboratory findings one gramme of hexamine was given four times a day to a patient without infection. To a sterile specimen of his urine a definite number of *Bacilli coli* were added, and plated after varying periods; 90% were killed in 15 minutes, 93% in one hour and 100% in two hours. The patient's urine had been several hours in his bladder before it was used as above and, therefore, an adequate splitting up of the hexamine had occurred. The practical result of the research is the conviction that, although hexamine is a valuable drug, its conversion into the necessary formaldehyde is slow and, therefore, the urine must be in contact with the diseased part for an hour or two in order to act. Hence the drug is of no value in its action on the kidney and even in the bladder it only acts slightly if frequency is at all a marked feature. The same reason explains the poor results when the catheter is tied in. Hexamine is valueless for urethral antisepsis, whatever the nature of the infection of the urethra.

(84) Hyperæsthesia of the Vesical Neck in Women.

C. G. Foote (*Urol. and Cutan. Review*, November, 1920) calls attention to the condition of hyperæsthesia of the neck of the bladder in women. Although in many instances a symptom of an organic condition, it is, in a greater number of cases, a neurosis. The pain in this neurotic form is associated with frequency of urination, and occurs either during micturition or, what is more common, after the act as an ache or discomfort. The treatment is simple and effective. Gradual dilatation of the urethra every three or four days, suggestion, regulation of diet and the use of cocaine in the urethra before dilatation when the hyperæsthesia is extreme. Cauterization of the neck of the bladder with silver nitrate is only rarely needed and is considered a *dernier ressort*.

British Medical Association News.

SCIENTIFIC.

A meeting of the Queensland Branch of the British Medical Association was held at the B.M.A. Rooms, Adelaide Street, Brisbane, on November 5, 1920, the President, Sir David Hardie, in the chair.

The President on behalf of the members of the Branch congratulated Dr. W. N. Robertson, who had been made a Commander of the Order of the British Empire in recognition of his services during the war.

On behalf of the Queensland members of the Australasian Medical Congress, Brisbane, 1920, the President of Congress, The Honourable W. F. Taylor, presented the Honorary General Secretary, Dr. A. Graham Butler, and the Honorary Co-adjutor Secretary, Dr. R. Marshall Allan, with a piece of plate each in recognition of their services.

Sir David Hardie, on behalf of the Council, presented Dr. R. Marshall Allan, the Honorary Secretary, with a silver cigarette box on the occasion of his approaching marriage.

Dr. A. Jefferis Turner moved:

That a deputation be appointed to interview the Home Secretary *re*

(*a*) the prevention of fly-borne disease and

(*b*) the use of lead paint.

He explained that this motion was being put to the meeting as a result of a resolution adopted at the Section of Diseases of Children at the Australasian Medical Congress, Brisbane, 1920. It was necessary for the Branch to move in the matter if effect were to be given to the resolution of Congress. The motion was seconded by Dr. D. Gifford Croll and carried. It was decided that the deputation should consist of the members of the Council, Dr. Jefferis Turner and Dr. J. Lockhart Gibson.

Dr. R. Graham Brown showed a specimen of epithelioma of the left vocal cord. The patient, a woman aged 53 years, had had a tumour of the left vocal cord for two years. It had been cauterized, in all, eleven times. There was no reaction to the Wassermann test and treatment with potassium iodide had been unsuccessful. When seen by Dr. Brown in May, 1920, the patient had an epitheliomatous growth on her left vocal cord and it was spreading towards the right cord. Preliminary tracheotomy and separation of the trachea from the œsophagus were performed and three weeks later a laryngectomy was done under rectal anæsthesia. The wound healed completely within three weeks. The pharynx was now clear except for a slight redness of the anterior wall and the patient could make fairly successful attempts at phonation.

Dr. Graham Brown also read notes on a case of aneurysm of the arch of the aorta and showed a radiogram in which the shadow of the aneurysmal dilatation was visible. The patient was a man of 58 years, who had been sent to him for an opinion on the condition of his throat. He had been a singer for years, but recently his voice had begun to "crack." He was losing weight and had a hard, dry cough of a somewhat brassy note. The throat was not sore and there was no difficulty in swallowing. There was no evidence of venereal infection. His wife was alive and well. On examination the intrinsic muscles of the left side of the larynx were found to be completely paralysed. There was also sufficient traumatic obstruction in the nose to account for the symptoms. The radiologist had reported the presence of a rarefying osteitis of the first rib, probably syphilitic in origin.

A discussion on cholecystitis was opened by Dr. E. D. Ahern, who dealt with the surgical aspect of the disease. Dr. G. P. Dixon discussed the method of incision for operations on the gall-bladder.

Dr. S. F. McDonald said that on one occasion in France he heard a discussion on the art of diagnosis by Sir John Rose Bradford, Sir Wilmot Parker Herringham and Sir George Makins. They all agreed that the diagnosis of pathological conditions of the abdomen, particularly in the right upper quadrant, was extremely difficult. Sir George Makins made the remark that "modern surgery had killed abdominal diagnosis." Dr. McDonald considered that certain points were very important for the correct diagnosis of gall-stones. In the first place, he was glad that Dr. Ahern had emphasized that the symptoms of cholecystitis lacked the definition and regularity which characterized those of gastric or duodenal ulcer. Secondly, rigors were a frequent feature of cholecystitis and in some cases were so severe that the disease resembled malaria. A third point of importance was the presence of cutaneous hyperæsthesia. Much of the work on this subject had been done by Sir James MacKenzie and further elaborated by David Ligat and Carmalt Jones. These observers had shown that for each abdominal viscus there was a corresponding cutaneous sensory area which often became acutely sensitive to touch when the viscus was inflamed. The area did not necessarily lie immediately superficial to the inflamed viscus. Ligat had recommended that in examining for the presence of hyperæsthesia, areas of skin should be successively pinched till the sensitive area was found. Carmalt Jones recommended the use of a pin to determine this area. It should be remembered that in nervous patients it was very easy to suggest hyperæsthesia when it was not really present. The hyperæsthetic skin area corresponding to disease processes of the gall-bladder lay immediately below the right costal margin in the skin supplied by the eighth and ninth dorsal nerves. Dr. McDonald described the pathology of gall-stones. They were composed of cholesterin, which was a product of all mucous surfaces and especially those which were inflamed. The cholesterin was deposited round a nucleus of calcium-bilirubin—a normal bile constituent which was precipitated when the bile-ducts were inflamed. Catarrh of the bile ducts could be due to an ascending infection from the duodenum or to the excretion of alimentary toxins into the bile by the liver cells. In either case inflammation was essential to the formation of gall-stones which became dissolved when introduced into a normal gall-bladder. Hence, in the treatment of gall-stones it was first of all necessary to correct any alimentary disturbance and so remove the focus of infection. In regard to treatment, the various solvents which had been recommended, had no effect on gall-stones *in vitro*, with the possible exception of olive oil, and, if given by mouth, none of them ever reached the gall-bladder. In his opinion, the best treatment for the condition was operation.

Dr. A. T. H. Nisbet described the radiographic diagnosis of gall-stones. He was of opinion that failure to detect them by radiographic means was no evidence of their absence and it was difficult to estimate exactly whether the diagnosis of their presence was correct, since a large number of patients concerned were never operated on. He had no doubt that by means of careful radiography gall-stones could be demonstrated in a large proportion of cases and when the diagnosis was doubtful, he was of opinion that the radiographer's assistance should be sought before a laparotomy was performed. Only a few days previously Dr. Higgins, of Townsville, had sent him a plate showing the shadow of a large gall-stone. The apparatus which had been used was a 30 cm. Cox's coil on a 65-volt circuit, which was bought ten years ago, and the time of exposure was 15 seconds. If by means of old apparatus biliary calculi could be demonstrated, surely good results in diagnosis could be obtained by the use of modern transformers with all their special advantages. Even in cholecystitis without stones in the gall-bladder the outline of the enlarged viscus could often be seen. He demonstrated this fact by means of a lantern slide. It should not be forgotten that a renal might be mistaken for a biliary calculus. Dr. Nisbet then showed six lantern slides in which gall-stone shadows were visible.

Dr. A. Jefferis Turner described the case of a young man whom he had seen in a military hospital in England. The patient suffered from intense abdominal pain and collapse, so that his condition suggested the diagnosis of a perforated viscus. Against perforation was the fact that the patient had had a rigor. Later, at operation, a number of gall-stones were removed. Dr. Turner raised the question as to whether gall-stones should always be removed by operation. Many women had come under his observation who enjoyed good health and suffered from colic at long intervals only. He did not believe in the use of solvents. A cholesterin-free diet might be of assistance in treatment. Liver, kidney, pancreas and eggs, especially the yolk, were the main articles of food which contained cholesterin. Some

of his patients while on a cholesterin-free diet had been free from attacks for some years.

Dr. D. A. Cameron mentioned a case of recurrence of gall-stones after operation. The recurrence had taken place in the common duct. The patient was a women aged 58, who had had attacks of jaundice for six years. In 1912 he had removed nine gall-stones from the common duct. In the winter of 1914 the patient had another attack of colic and Dr. Cameron operated, removing a large gall-stone from the ampulla of Vater. The attacks of colic recurred in 1915. Another operation was performed in August, 1918, and five stones were removed from the common duct. The gall-bladder was small, shrivelled and functionless. It was bound down by masses of adhesions and no attempt was made at removal.

The patient had since been free from colic. For the purposes of cholecystectomy Dr. Cameron preferred Hannam's table to an ordinary operating table and a pad placed under the patient's back. Cholecystectomy was a more difficult procedure than cholecystostomy and was associated with a greater risk of serious hæmorrhage. The exhibition of calcium salts or the transfusion of blood was not always successful in treating this complication. After cholecystectomy any stones present in the common duct should be removed and the surgeon should be quite sure that the duct was patent. It was advisable to pass Hegar's dilators and if this were done it was unnecessary to drain the duct. If the attack were acute and operation decided on, the gall bladder should be merely drained. In chronic cases the gall bladder should be removed. The bile duct should be dilated and its proximal end securely closed.

Dr. J. A. Cameron called the attention of members to a paper by G. E. Waugh in a recent issue of the *British Journal of Surgery* on the subject of mobile ascending colon. This condition resembled in its clinical manifestations inflammatory conditions of the gall-bladder and was diagnosed by a consideration of the symptoms and the radiographic findings. The laity should be persuaded to undergo immediate operation when troubled with gall-stones. Incisional hernia after gall-bladder operations had, in Dr. Cameron's experience, only resulted when a vertical incision was made. He preferred the Kocher incision and the patient was always placed in the reversed Trendelenberg position. He had also tried the Perthes incision, which gave good exposure, though it took a longer time to make and to close. Cholecystectomy was more risky than drainage of the gall-bladder because of the frequency of abnormality of the cystic duct and artery.

Dr. John Jackson drew attention to the work done at the Mayo Clinic on the functions of the gall-bladder. He objected to the statement that infection travels through the common duct from the duodenum. Infection through the lymphatics was an important factor. Rosenow had proved that infection was hæmatogenous and due to the streptococcus in nearly all cases.

Dr. L. M. McKillop said that he favoured removal of the gall-bladder whenever possible, for three reasons: Firstly, if the gall-bladder were left behind, there was a chance of recurrence of the gall-stones; secondly, cholecystectomy was associated with no more risk than cholecystostomy, if the operation were carefully performed; and, lastly, once the gall-bladder had become infected, it could no longer be trusted. It was liable to undergo chronic pathological changes which predisposed to the formation of calculi in the cystic or common duct. The surgeon should make sure that the common duct was patent before he closed the incision. The incision Dr. McKillop favoured was division of the rectus muscle half way between the ensiform cartilage and umbilicus. In regard to diagnosis, a radiographic plate showing the presence of gall-stones was important, but much more valuable was the rapid emptying of the stomach within two hours or less after the administration of a bismuth meal. He emphasized the intolerance of patients suffering from cholecystitis to foods containing fats and considered this an important point in the diagnosis.

Dr. E. S. Meyers referred to the difficulties in diagnosis and mentioned a case in which the patient's only symptom was constant epigastric pain. A diagnosis of chronic appendicitis was made, but at operation the appendix was found to be normal and a stone was located in the cystic duct. The overlooking of calculi was due mainly to insufficient exposure of the field of operation.

Dr. V. McDowall agreed with Dr. J. A. Cameron that at bismuth meal examinations it was common to find the ascending colon in unusual positions. Only a small percentage of gall-stones contained salts which were opaque to the X-rays. The absence of a gall-stone shadow was of no importance. It was hard to differentiate between duodenal ulcer, inflamed gall-bladder, chronic appendicitis and adhesions.

Dr. E. D. Ahern stated that organisms were found in the wall of the gall-bladder and not in its contents. Only once in 15 cases were organisms found in the bile. Rigors occurred more commonly in cholelithiasis than in cholecystitis. In regard to the jaundice associated with the presence of gall-stones, he believed in the inflammatory theory of their causation rather than the mechanical. He agreed with Dr. McDowall that failure to find shadows of calculi by radiography was not of great importance. He quoted the percentage of cure of patients under the care of a well-known American surgeon who claimed a successful result in 90% of cases of gall-stones and 46% of cases of cholecystitis. This showed the need for improvement in the method of dealing with the latter condition.

MEDICO-POLITICAL.

A meeting of the Victorian Branch was held at the Medical Society Hall, East Melbourne, on February 2, 1921, Dr. Basil Kilvington, the President, in the chair.

The Honorary Treasurer, Dr. C. H. Mollison, presented the financial statements of the Victorian Branch and of the Medical Society of Victoria for the year ended January 13, 1921. After explaining the several items in brief, he moved the adoption of the statement. The motion was carried.

The financial statements will be found on page 182.

The undermentioned have been nominated for election as members of the New South Wales Branch:

Wilfred Lievesley Nickson, Esq., M.B., Ch.M., 1920 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Charles V. W. Brown, M.B., Ch.M., 1920 (Univ. Syd.), "Wyncroft," 104 Alfred Street, North Sydney.

The undermentioned have been elected as members of the New South Wales Branch:

R. B. Austin, Esq., M.B., Bac. Surg., 1910 (Univ. Glasg.), Armidale.

J. R. Broome, Esq., M.B., Ch.M., 1920 (Univ. Syd.), Cronulla.

F. J. Gwynne, Esq., M.B., Ch.M., 1920 (Univ. Syd.), 265 Elizabeth Street, Sydney.

J. Hughes, Esq., M.B., Ch.M., 1920 (Univ. Syd.), Cessnock.

R. C. Traill, Esq., M,B.. Ch.M., 1920 (Univ. Syd.), Clarence Street, Burwood.

The undermentioned has been elected a member of the Queensland Branch:

Harold William Savige, M.B., B.S. (Univ. Melb., 1918).

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

The undermentioned have been registered, under the provisions of the *Medical Acts, 1912 and 1915*, as duly qualified medical practitioners:

Browne, Denis John, M.B., 1914, Univ. Sydney (England).

Dunsmuir, Allan, M.B., Bac. Surg., 1907, Univ. Glasgow (Narrandera).

Hills, George Ridge, M.B., Bac. Surg., 1920, Univ. Adelaide (Broken Hill).

Rieger, Clarence Oscar Ferrero, M.B., Bac. Surg., 1919, Univ. Adelaide (Broken Hill).

Steven, Arthur Raphael, M.B., Bac. Surg., 1916, Univ. Glasgow (Gladesville).

Symons, Eric Lindsay, M.B., Bac. Surg., 1917, Univ. Adelaide (Broken Hill).

Watson, Sara Adam, now Youdale, Sara Adam, M.B., Bac. Surg., 1914, Univ. Glasgow (Burwood).

The following change of name has been registered:
Grace Marion Cordingley to Grace Marion Bridge (Nowra).

VICTORIA.

The undermentioned has been registered under the provisions of Part I. of the *Medical Act, 1915*, as a duly qualified medical practitioner:
Harold David Bowyer Miller, M.B., B.S., Melb., 1919 (Maryborough).
The name of the late Dr. William Morrison has been removed from the Register.

On another page in this issue will be found an advertisement calling for applications for the positions of Tata Professor of Clinical Medicine and Therapy and of Tata Professor of Protozoology at the School and Hospital of Tropical Medicine to be established under the auspices of the Indian Government at Bombay. Unfortunately the information reached us very late. Medical practitioners in Australia desiring to apply for either of these important positions, should send a preliminary application by cable to the Secretary of the Royal Society, Burlington House, London, W.. Week-end cables can now be sent to England by the Pacific Cable Board.

BRITISH MEDICAL ASSOCIATION (VICTORIAN BRANCH).

Revenue and Expenditure Account for Period From January 15, 1920, to January 13, 1921.

Expenditure.

	£	s.	d.
To Medical Society of Victoria	1,613	17	9
„ *British Medical Journal*	1,045	9	0
„ Postages, Duty Stamps	46	1	4
„ Printing	44	17	0
„ Stationery	40	11	10
„ Clerical Assistance	2	1	0
„ Commission (New Members)	2	10	0
„ Advertising	8	15	10
„ Audit for 1919	2	2	0
„ Travelling Expenses		16	5
„ Rebates to Divisions (2 Years)	71	2	0
„ Sundries and Telephone Calls	8	16	0
„ Post-graduate Classes	21	1	3
„ Bank Charges	4	4	5
„ Electrical Replacements	1	10	6
„ Balance	304	0	7
	£3,217	16	11

Revenue.

	£	s.	d.	£	s.	d.
By Balance, January 15, 1920				349	17	10
„ Sales of Books, etc.				17	12	10
„ Subscription—						
Metropolitan Members	£1,800	6	6			
Country Members	895	1	3			
Junior Members	164	6	6			
Rebate (London)	1	2	0			
	£2,860	16	3			
Less Refund	10	10	0			
				2,850	6	3
				£3,217	16	11

Liabilities.

	£	s.	d.
To Balance as above	304	0	7
	£304	0	7

Assets.

	£	s.	d.
By Bank of Victoria, Ltd.	192	9	1
„ Commonwealth Bank of Aust. (Balance)	8	11	6
„ Trustees, Organization Fund	100	0	0
„ Petty Cash in Hand	3	0	0
	£304	0	7

Audited and found correct,
J. V. M. WOOD & CO., F.I.C.A., Auditors.
Melbourne, February 10, 1921.

C. STANTON CROUCH, Secretary.
C. H. MOLLISON, Honorary Treasurer.

MEDICAL SOCIETY OF VICTORIA.

Revenue and Expenditure Account for Period From January 15, 1920, to January 13, 1921.

Expenditure.

				£	s.	d.
To *The Medical Journal of Australia*				952	7	10
„ Salaries—						
Secretary	£412	0	0			
Library Clerk	41	0	0			
Caretaker	26	0	0			
Assistant	35	4	2			
				514	4	2
„ Library Journals and Binding				29	4	4
„ Postages, etc.				46	0	0
„ General Expenses				2	13	8
„ Rates, Taxes and Insurance				14	17	5
„ Telephone				16	13	2
„ Light and Power				9	5	4
„ Audit for 1919				3	3	0
„ Bank Charges				1	0	0
„ Repairs				20	11	6
„ Office Furniture				20	4	0
„ Balance in Bank of Victoria, Ltd.				113	10	3
				£1,743	14	8

Revenue.

	£	s.	d.
By Balance, January 15, 1920	129	16	11
„ Subscriptions, 1920	1,613	17	9
	£1,743	14	8

Audited and found correct,
J. V. M. WOOD & CO., F.I.C.A., Auditors.
Melbourne, February 10, 1921.

C. STANTON COUCH, Secretary.
C. H. MOLLISON, Honorary Treasurer.

Correspondence.

DIGESTIBILITY OF RAW CORN STARCH.

Sir: In the issue of this *Journal* for January 29, 1921, on page 95 appears a note on the above.

In my final year at St. Thomas's (1881) I remember a recently qualified man coming back after a voyage to the West Indies, expatiating on the value of raw arrowroot for acute choleraic diarrhœa. A teaspoonful of raw arrowroot stirred into a wine-glassful of cold milk worked wonders, he assured us, repeated every three hours, if required. I made a mental note of that and have tried the method on many occasions with strikingly beneficial results.

As far as one could tell the method was a purely empirical one. The researches of Messrs. Langworthy and Deuel (as given in the *Journ. Biolog. Chemistry*, March, 1920) show that like many other empirical observations the suggestion has a scientific basis.

The copious watery discharges of the acute dysenteric or choleraic diarrhœa are a big drain on the strength of the patient, often leading to fainting and collapse. The absorption of the whole of the starch thus given helps to counteract the drain and leads to a rapid reinforcement of the digestive secretions and ferments of the alimentary canal, restoring the normal balance that has been overcome by the exciting causes of the diarrhœa.

Yours, etc.,
LEONARD W. BICKLE, F.R.C.S. (Edin.).

Sydney,
February 8, 1921.

THE HEALTH OF SCHOOL CHILDREN IN QUEENSLAND.

Sir: The children are examined at three stages of their school career:

(*a*) At entry.
(*b*) At nine years.
(*c*) About to leave.

This accounts somewhat for numbers as quoted. In regard to remedial measures, there is a follow-up system, pointing out to parents what is wrong with the child and the course to pursue. I have always advised the teachers to inform parents that the general hospital is ready to treat children whose parents cannot afford the private practitioner. This has been readily availed of. A large proportion of the defects is comprised of tonsils and adenoids and it has become necessary or, rather, convenient to set a morning apart for these. I find that teachers are keen on keeping the parents up to the mark in regard to treatment and it is but rarely that one finds on a second examination "nothing done."

In conclusion I must state that it would be hard to find a set of more sturdy and healthy children.

Yours, etc.,
R. FRESHNEY.

Toowoomba, Queensland,
January 28, 1921.

THE VENEREAL DISEASES ACT IN NEW SOUTH WALES.

Sir: With regard to the performance by medical practitioners of the requirements of the *Venereal Diseases Act, 1918* (New South Wales), I should like to point out a few facts, which would, I think, tend to counteract some of tendency to evade performance of these requirements shown by many medical practitioners.

One of the most frequent objections given is that a strict insistence on notification will drive a medical man's patients from him to some other whose conscience is more elastic. This objection, I think, is grossly over-rated, as I find that the practice of writing out the notification form in the presence of the patient and handing it to him to show that there is no means of identifying him with the information contained therein, except by reference to the butt retained in my book, immediately sets his mind at rest.

The next is that the difficulty of keeping check on the patient's attendance renders it almost impossible for a busy practitioner to comply with the requirements. But if an appointment book is kept and a special mark, such as a big V, set against the name, failure to keep that appointment will remind one that a note of warning has to be dispatched and the particular case card kept in a prominent place as a reminder that further action may be required. The amount of correspondence which may become necessary, is certainly a trial, but if on change of medical adviser the new man would write on the proper form, giving his identification number for the case, much is saved. Instead, a personal note is sometimes sent, failing to give this necessary information and another letter required by the failure of the medico and not by the fault of the Act.

But in spite of disadvantages and trials which await the practitioner, it still remains a fact that this Act confers a considerable benefit upon the individual medico and the profession as a whole.

The latter is assured the sole right to treat such cases and when treated privately this means a considerable sum diverted into the possession of our profession—when one considers how large a proportion were treated by people outside the profession before the passage of the Act.

The individual practitioner is assured of this, that, having commenced treatment of a case, he is able to insist upon completion of the treatment and the corresponding fees, instead of losing his patient after the first couple of doses of arseno-benzene has obliterated the lesion for which the patient applied for treatment. If every practitioner insists upon notifying, there would be little or no advantage to the patient to fly from one medico to his rival.

If the rival fails to notify a case which comes to him from another practitioner, he can be forced to do so by the first man insisting on receipt of form A., which makes the newcomer give his identification number of the case. This is then transmitted to the Commissioner on form D. by first man.

If no such identification number is reported by the rival, then the Commissioner will no doubt take the necessary steps and the rival's advantage will be gone.

But in addition to all this there is the desirability of testing properly this means of coping with an immensely serious problem and the duty of the members of the medical profession to obey the law and to refrain from attempting to take a supposed advantage over fellow practitioners.

If the moral obligation does not appeal to all, I think the first-mentioned pecuniary advantage should be efficacious if well understood in inducing all men to notify such cases.

Yours, etc.,
E. H. MOLESWORTH.

"Beanbah,"
235 Macquarie Street, Sydney.
(Undated.)

WOMAN SCHOOL MEDICAL OFFICER FOR EGYPT.

Sir: I shall be glad to hear from any woman graduate in medicine who will contemplate school inspection work in Egypt. The post available is Assistant Medical Officer. The requirements are: Age about 30 years; some knowledge of eye work and school work desirable. The salary offered is £540 per annum, with allowances, and the appointment will be ratified by a contract.

Yours, etc.,
JAMES W. BARRETT.

105 Collins Street, Melbourne,
February 17, 1921.

SUPERVISION AND REGISTRATION OF MIDWIVES.

Sir: Since the appearance of Dr. Purdy's paper in your columns, correspondents have written, giving their views with respect to the supervision and registration of midwives.

While I am quite in sympathy with their views, I have been struck by the fact that none of them focussed attention on reforms that are at present practicable. The time is not yet ripe to eliminate "Gamp," but surely the time

has come when the Department of Public Health should refuse to continue her in charge of a registered private hospital. Yet in some of our country towns, even where there is a registered private hospital run by a certificated nurse, we find that she is undercut by an untrained and uncertificated woman who has registered her dwelling as a private hospital and has taken up midwifery work. This is an abuse that could be very easily stopped, as the matter is entirely in the hands of the Director of Public Health.

Another reform, at present practicable, although not quite so easily carried out, is a law or regulation that would prevent unregistered women from inserting "ads." in the local papers that would lead any ordinary reader to infer that they are duly trained and registered obstetric nurses.

If the members of our profession throughout the State[1] make any serious effort to secure such reforms as are at present practicable, we should be able to secure the first of the reforms suggested above, at once, and the second, very soon.

Yours, etc.,
"COUNTRY PRACTITIONER."

Medical Appointments.

The appointment as Resident Medical Officers of Drs. K. S. Hetzel, S. R. Hecker (B.M.A.), F. R. Hone, S. E. Francis, W. E. Steven, R. L. E. Walmsley, H. G. Wallace and Bertram E. Wurm at the Adelaide Hospital Department is announced in *The South Australian Government Gazette.*

Dr. C. H. Bennett has been appointed Junior Resident Medical Officer at the Perth Public Hospital.

It is announced that Dr. E. F. O'Sullivan has been appointed District Medical Officer and Public Vaccinator at Mornington, Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xxiii.

University of Melbourne: Demonstrators in Histology.

Bombay School of Tropical Medicine: Tata Professors of Clinical Medicine and Therapy and of Protozoology.

Alfred Hospital, Melbourne: Various Vacancies on the Medical Staff.

Croydon District Hospital, North Queensland: Medical Officer.

Medical Appointments.

IMPORTANT NOTICE.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429 Strand, London, W.C..

Branch.	APPOINTMENTS.
NEW SOUTH WALES. (Hon. Sec., 30-34 Elizabeth Street, Sydney.)	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIA. (Hon Sec., Medical Society Hall, East Melbourne.)	All Institutes or Medical Dispensaries. Manchester Unity Independent Order of Oddfellows. Ancient Order of Foresters. Hibernian Australian Catholic Benefit Society. Grand United Order of Free Gardeners. Sons of Temperance. Order of St. Andrew. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND. (Hon. Sec., B.M.A. Building, Adelaide Street, Brisbane.)	Australian Natives' Association. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIA. (Hon. Sec., 3 North Terrace, Adelaide.)	Contract Practice Appointments at Renmark. Contract Practice Appointments in South Australia.
WESTERN AUSTRALIA. (Hon. Sec., 6 Bank of New South Wales Chambers, St. George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND: WELLINGTON DIVISION. (Hon. Sec., Wellington.)	Friendly Society Lodges, Wellington, New Zealand.

[1] Our correspondent refers to the State of New South Wales.

Diary for the Month.

Mar. 1.—N.S.W. Branch, B.M.A., Ethics Committee; Executive and Finance Committee.
Mar. 2.—Vic. Branch, B.M.A..
Mar. 3.—N.S.W. Branch, B.M.A., last day for nomination of candidates for election to the Council.
Mar. 4.—Q. Branch, B.M.A..
Mar. 8.—Tas. Branch, B.M.A..
Mar. 8.—N.S.W. Branch, B.M.A.. Medical Politics Committee; Organization and Science Committee.
Mar. 11.—Q. Branch, B.M.A., Council.
Mar. 11.—S. Aust. Branch, B.M.A., Council.
Mar. 15.—N.S.W. Branch, B.M.A., Council (Quarterly).
Mar. 16.—W. Aust. Branch, B.M.A..
Mar. 17.—Vic. Branch, B.M.A., Council.
Mar. 17.—N.S.W. Branch, B.M.A., last day for return of ballot papers for election of the Council.
Mar. 18.—N.S.W. Branch, B.M.A., Annual General Meeting.

EDITORIAL NOTICES.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned.

Original articles forwarded for publication are understood to be offered to *The Medical Journal of Australia* alone, unless the contrary be stated.

All communications should be addressed to "The Editor," *The Medical Journal of Australia*, B.M.A. Building 30-34 Elizabeth Street, Sydney. (Telephone: B. 4625.)